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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/676,248	09/30/2003	Peter K. Rogan	33026	5913

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EXAMINER	
POHNERT, STEVEN C	
ART UNIT	PAPER NUMBER
1634	

SHORTENED STATUTORY PERIOD OF RESPONSE	MAIL DATE	DELIVERY MODE
3 MONTHS	03/27/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

Office Action Summary	Application No.	Applicant(s)	
	10/676,248	ROGAN ET AL.	
	Examiner	Art Unit	
	Steven C. Pohnert	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *16 January 2007*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final..

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-42* is/are pending in the application.

4a) Of the above claim(s) *1-32* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *33-42* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *30 September 2003* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

This action is in response to the papers filed on 1/16/2007. Currently, claims 1-42 are pending. Claims 1-33 have been withdrawn. Claims 34-42 have received action on the merits. All arguments have been thoroughly reviewed but are not deemed persuasive for the reasons that follow.

This action is FINAL

Any rejections not reiterated below are hereby withdrawn in view of amendments and arguments.

a. The 112, 2nd paragraph over "less than about" have been withdrawn in view of the amendments to the claims

b. The 112, 2nd paragraph over paralogous genes has been withdrawn in view of the arguments on page 10 of the 1/16/2007 response.

Grounds for New Rejection

Claim Rejections - 35 USC § 112

1. The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Newly amended claims 34-36 and 41 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Newly amended claims 34-36 and 41 recite, "having a condition selected from the group consisting of idiopathic mental retardation, mental retardation and at least one other clinical abnormality, mental retardation and cancer, and combinations thereof." It is unclear if the claim requires the condition of mental retardation and cancer, or idiopathic mental retardation and mental retardation (idiopathic mental retardation is encompassed by mental retardation), or idiopathic mental retardation and cancer. If applicants intend to require idiopathic mental retardation or mental retardation and at least one other clinical abnormality, mental retardation and cancer, and combinations thereof, applicants may wish to add an "or" between the first two conditions.

Maintained rejections

Claim Rejections - 35 USC § 102

2. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

3. Claims 34-37 and 41 are rejected under 35 U.S.C. 102(b) as being anticipated by Rogan, et al (Genome Research, 2001, volume 11, pages 1086-1094).

With regards to claim 34, Rogan et al teaches a method to design and produce custom 2-kb to 10 kb genomic single copy probes to detect genetic rearrangements and common chromosome abnormalities (see page 1086, lines 4-19). Rogan et al further teaches probes to chromosomal regions 15q11.2, 22q11.2, and 1p36 (see page 1091, 1st paragraph of discussion). Rogan et al teaches 22q11.2 probes demonstrate a deletion only in cells from a DiGeorge's patient. Rogan thus teaches methods of screening individuals with DiGeorge syndrome, which is associated with mental retardation, with probes to detect cytogenetic abnormalities (claim 34) (see 1090 column 2, lines 1-8 and figure 4).

With regards to claim 35, Rogan teaches the 22q11.2 probe hybridizes to both copies of chromosome 22 in control cells, but only 1 chromosome in the DiGeorge Syndrome individual (page 1090, column 2, lines 11-13). Thus the absence of hybridization of the 22q11.2 probe to chromosome 22 associates a hybridization pattern with a specific clinical abnormality.

With regards to claim 36, Rogan teaches single copies probes (see figure 1 and page 1086, column 2 lines 23-24).

With regards to claim 37, Rogan et al teaches a method to design and produce custom 2-kb to 10 kb genomic single copy probes to detect genetic rearrangements and common chromosome abnormalities (see page 1086, lines 4-19). Rogan et al further teaches probes to chromosomal regions 15q11.2, 22q11.2, and 1p36 (see page 1091, 1st paragraph of discussion). Rogan et al teaches 22q11.2 probes demonstrate a deletion only in cells from a DiGeorge's patient. Rogan thus teaches methods of

screening individuals of DiGeorge syndrome (known clinical abnormalities) with probes to detect cytogenetic abnormalities (claim 34) (see 1090 column 2, lines 1-8 and figure 4). The absence of hybridization of 22q11.2 to chromosome 22 demonstrates a chromosome lacks this region and is thus imbalanced.

With regards to claim 41(this rejection is necessitated by amendment), Rogan teaches probes to 1p36 which is within 8,000 kb of the end of the chromosome.

Response to Arguments

4. The response filed 1/16/2007, asserts on page 11, that the claims require that the sequence and location of the probes of the instant invention are known, while the size and sequence of the nick-translated probe from the Rogan is unknown.

Applicant's arguments have been fully considered but are not found persuasive.

With respect to the arguments directed to the size and sequence of the probes of Rogan being unknown, Rogan et al teaches the probes were identified from the HIRA, MAGEL2, and CDC2L1 genomic sequences (see page 1087, column 1). Rogan et al further teaches the probes from MAGEL2 are 4100 bp, 3544 bp, and 2290 bp in length (see 1087, 2nd column, 1st paragraph), which is les than 25 kb. Rogan et al further teaches CDC2L1 has probes of 4823 bp, and 4724 bp (see 1087, 2nd column, 1st paragraph), beginning with intron 11 and ending with the 3'UTR (see page 1088, 1st column, 1st paragraph), which is les than 25 kb. The HIRA, MAGEL2 and CDC2L1 sequences are known. Rogan et al further teaches the Rogan et al further teaches the length of probes to the HIRA locus, lengths, and the relation to introns and exons (see 1087, 2nd column, last paragraph). Rogan et al teaches the 32544 bp probe of MAGEL2

began 204 bp downstream the first coding nucleotide of and contained 1789 bp of expressed coding (see 1087, 2nd column, last paragraph). Rogan et al thus does teach probes of known length and sequence. Rogan teaches where the probes interact with introns, exons and coding sequences giving an exact genomic location. Further the response asserts that the nick translation results probes of unknown length, however the claim requires only that the probes have a length of less than 25 kb. Thus the probes of Rogan meet the limitations of the claims. Thus rejections based on the work of Rogan et al are maintained.

5. Claims 34-42 are rejected under 35 U.S.C. 102(b) as being anticipated by Flint, et al (Nature Genetics, 1995, volume 9, pages 132-140).

With regards to claim 34, Flint et al teaches fluorescence in situ hybridization (FISH) labeled by nick translation (see page 139 first column fluorescence in situ hybridization) and detection of a deletion of 13q region in patient AH, while no deletion was found in parent (see page 133, 2nd column lines 19-24and figure 2b). Patient AH has idiopathic mental retardation (page 133, column 1 line 8). Nick translation results in multiple probes of less then 25 kb. The deletion taught by Flint demonstrates a chromosome 13q imbalance, which is a cytogenetic abnormality.

With regards to claim 35, Flint teaches detection of a deletion of 13q region in patient AH, while no deletion was found in parent (see page 133, 2nd column lines 19-24and figure 2b). Flint teaches patients examined have idiopathic mental retardation

(page 133, column 1 line 8). Thus the 13q chromosomal imbalance is a cytogenetic abnormality associated with a specific clinical abnormality.

With regards to claim 36, Flint teaches the D13S107 hybridizes to the 13q arm. Flint thus teaches a single hybridization signal from a plurality of probes to a single chromosomal.

With regards to claim 37, Flint et al teaches fluorescence in situ hybridization (FISH) labeled by nick translation (see page 139 first column fluorescence in situ hybridization) and detection of a deletion of 13q region in patient AH, while no deletion was found in parent (see page 133, 2nd column lines 19-24and figure 2b). Nick translation results in multiple probes of less then 25 kb. The deletion taught by Flint demonstrates a chromosome 13q imbalance.

With regards to claim 38, Flint teaches patients examined have idiopathic mental retardation (page 133, column 1 line 8). As patient AH has idiopathic mental retardation and the deletion detected at 13q, is thus indicative of idiopathic mental retardation. Flint thus teaches the step of correlating chromosomal imbalances to a specific medical condition.

With regards to claim 39, Flint teaches use of nick translated probes, which result in a plurality of probes.

With regards to claim 40, Flint teaches the D13S107 hybridizes to the 13q arm. Flint thus teaches the hybridization of a plurality of probes to a specific chromosomal arm.

With regards to claims 41 and 42 (this rejection is necessitated by amendment), Flint further teaches the four probes including globin 3' HVR, p157.9, pYNZ32 and MS600 that have been physically localized to 170 kb from the 16p telomere, p157.9 and pYNZ32 1.6mB (1600kb) and 2.2 Mb (2,200 kb) from the 4p and MS600 70-80 kb from the pseudoautosomal telomere (see page 138, 2nd column, last full paragraph). Flint thus teaches probes detecting cytogenetic abnormalities 8,000 kb or less from the end of the chromosome.

Response to Arguments

6. The response filed 1/16/2007, asserts on page 12, that the claims require that the sequence and location of the probes of the instant invention are known, while the size and sequence of the nick translated probe from the Flint is unknown.

Applicant's arguments have been fully considered but are not found persuasive.

Flint further teaches that four probes including globin 3' HVR, p157.9, pYNZ32 and MS600 that have been physically localized (see page 138, 2nd column, last full paragraph). Physically localized is directed to a specific sequence and location. Further Flint et al teaches in table 1, the probe names and the restriction enzyme by which the probe was excised from the vector of propagation (see table 1, page 133). The teaching by Flint of the restriction site required to excise the probe, inherently teaches the sequence of the probes, as the artisan would thus be able to determine the sequences of the restriction site, and thus the sequence of the probe. It is further noted that the claims do not require the entire probe sequence to be known, thus the teachings of Flint meet the known sequence argument. Further Flint teaches the

MS626 probe was excised from a plasmid (see page 133, 2nd column, lines 26-30), the use of a probe from a plasmid results in a probe of smaller than 100 kb recited in the response, further the probes are synthesized by nick translation resulting in probes on less than 25 kb. Thus hybridization of this probe would result a distinct location. Further the response asserts that the nick translation results probes of unknown length, however the claim requires only that the probes have a length of less than 25 kb. Thus the probes of Flint meet the limitations of the claims.

7. Claims 34-40 are rejected under 35 U.S.C. 102(b) as being anticipated by Bentz et al (Blood, 1994, volume 83 pages 1922-1928).

With regards to claim 34, Bentz teaches hybridization of nick translated YAC-probe D107F9, results in 2 hybridization signals from normal cells and 3 signals from BCR-ABL cells (see page 1923, column 2, lines 1-4and figures 1 and 2). Nick translation results in a plurality of short probes, all less then 25 kb. Bentz further teaches the 2 signals in normal cells are due to hybridization to chromosome 22 (see page 1923, column 2, lines 4-6 and figures 1) and the third signal is due to a translocation of chromosome 22 to chromosome 9q. Bentz teaches the BCR-ABL translocation detected by the D107F9 is indicative of CML or Ph-positive ALL (see abstract). Bentz thus teaches a method of screening individuals with cancer for cytogenetic abnormalities using a probe of less then 25 kb. The hybridization pattern of the probes is indicative of cytogenetic abnormalities.

With regards to claim 35, Bentz teaches the D107F9 probe hybridization detects an imbalance in both chromosome 22 and 9 of the BCR-ABL positive cells (see page

1923, column 2, lines 1-4and figures 1 and 2). These imbalances are associated with ALL and CML, which are specific clinical abnormalities.

With regards to claim 36, Bentz teaches the D107F9 probe hybridization detects chromosome 22 of normal cells. (See page 1923, column 2, lines 4-6 and figures 1)

With regards to claim 37, Bentz teaches hybridization of nick translated YAC-probe D107F9, results in 2 hybridization signals from normal cells and 3 signals from BCR-ABL cells (see page 1923, column 2lines 1-4and figures 1 and 2). Nick translation results in a plurality of short probes, all less then 25 kb. Bentz further teaches the 2 signals in normal cells are due to hybridization to chromosome 22 (see page 1923, column 2 lines 4-6 and figures 1) and the third signal is due to a translocation of chromosome 22 to chromosome 9q. Bentz teaches the BCR-ABL translocation detected by the D107F9 is indicative of CML or PC-positive ALL. Bentz thus teaches a method of detecting chromosome imbalances using a probe of less then 25 kb to determine imbalances.

With regards to claim 38, the D107F9 probe detects an imbalance in both chromosome 22 and 9 of the BCR-ABL positive cells (see page 1923, column 2, lines 1-4and figures 1 and 2). These imbalances are associated with ALL and CML, which are cancers.

With regards to claim 39, the nick translated D107F9 probe is a plurality of probes.

With regards to claim 40, the D107F9 is specific to chromosome 22q11, which is a specific chromosome arm. Flint thus teaches the hybridization of a plurality of probes to a specific chromosomal arm.

Response to Arguments

8. The response filed 1/16/2007, asserts on page 12, that the sequence and location of the probes of the instant invention are known, while the size and sequence of the nick translated probe from the Bentz is unknown. The response further asserts that the specificity of the probes is limited to the 215 kb usually contained in a YAC construct.

Applicant's arguments have been fully considered but are not found persuasive.

Further the assertion that Bentz's teaching are limited to the detection of fragments of 215 kb usually encompassed by a YAC, Bentz teaches the use of PCR primers CL1 and CL2 to obtain fragments of the YAC (see page 1923, 1st column, lines 5-8). Thus Bentz does not teach the use of 215 kb fragments. Further as the sequence of the CL1 and CL2 are disclosed by Bentz the sequence of the probes are known. It is noted that the claims do not require the entire sequence be known. Further Bentz teaches the use of a fragment from the 3' coding and 3' flanking sequences of the human ABL gene (see page 1923, 1st column, lines 15-18). The sequence of this fragment inherently must be known, so as to be able to describe the probe as corresponding to the coding and flanking sequences. Further the response asserts that the nick translation results probes of unknown length, however the claim requires only

that the probes have a length of less than 25 kb, the nick translation of Bentz results in probes of less than 25 kb. Thus the probes of Bentz meet the limitations of the claims.

Double Patenting

9. The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

10. Claims 34-37 and 39-40 are rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1 and 3 of U.S. Patent No. 7014997. Although the conflicting claims are not identical, they are not patentably distinct from each other because they are co-extensive in scope.

Claim 34 of instant application is drawn to a method of screening individuals with clinical abnormalities with a plurality of probes. The hybridization of said probes resulting in patterns indicative of cytogenetic abnormalities. Claim 3 of '997 patent

teaches the detection of hybridization pattern for detection of cytogenetic abnormalities. Claim 1 of '997 patent teaches chromosome abnormalities are indicative of pathological abnormalities.

Claim 35 of instant application is drawn to associating hybridization patterns of probes with clinical abnormalities. Claim 1 of '997 patent teaches hybridization is indicative of pathological conditions.

Claim 36 of instant application is drawn to probes hybridizing to a single genomic location. Claim 1 of '997 patent teaches a nucleic acid probe complementary to a non-repetitive portion of genome. A non-repetitive portion of the genome would result in probes hybridizing to a single genomic location.

Claim 37 of instant application is drawn to detecting and delineating the extent of chromosome imbalances by comparison of probe hybridization to a standard genome map. Claim 1 of '997 patent teaches hybridization of nucleic acid of non-repetitive sequence probes with known genomic sequence coordinates. The hybridization of probes from claim 1 of '997 patent detect chromosome imbalances and since known genomic coordinates are known to delineate extent by comparison to standard genomic map.

Claim 39 of instant application is drawn to a method of utilizing a plurality of probes. Claim 1 of '997 patent teaches the use of a pair of probes, which is a plurality.

Claim 40 of instant application is drawn to detecting and delineating the extent of chromosome imbalances by comparison of probe hybridization to a standard genome map. Claim 1 of '997 patent teaches hybridization of nucleic acid of non-repetitive

sequence probes with known genomic sequence coordinates. The hybridization of probes from claim 1 of '997 patent detect chromosome imbalances and since known genomic coordinates are known the specific chromosome arm is also known.

Response to Arguments

11. The response filed 1/16/2007, asserts on pages 13-14, that the probes of the present invention are drawn to detection of cytogenetic abnormalities associated with idiopathic mental retardation, mental retardation, cancer or combinations thereof, while the claims of '977 are drawn to pathological conditions. The response further asserts that the probes of the instant application are drawn to the "subtelomeric region".

Applicant's arguments have been fully considered but are not found persuasive.

While the current claims are drawn to cytogenetic abnormalities associated with idiopathic mental retardation, mental retardation, cancer or combinations thereof, these are pathological conditions. Thus the claims of '977 encompassed by the instant claims.

For the amended claim 37 recite "a subtelomeric region of a chromosome", however the specification does not specifically define what the subtelomeric region encompasses. Thus SEQ ID No 1-428 and 447-479 of claim 1 of '977 are directed to subtelomeric regions of the chromosome.

12. Claim 38 is rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claim 1 of U.S. Patent No. 7014997 in view of Bentz et al (Blood, 1994, volume 83 pages 1922-1928). Claim 1 of '997 patent teaches hybridization of nucleic acid of non-repetitive sequence probes with known

genomic sequence coordinates. The hybridization of probes from claim 1 of '997 patent detect chromosome imbalances and since known genomic coordinates are known to delineate extent by comparison to standard genomic map. Claim 1 does not teach correlating imbalances with medical conditions including cancer.

However, Bentz teaches the D107F9 probe hybridization detects an imbalance in both chromosome 22 and 9 of the BCR-ABL positive cells (see page 1923, column 2, lines 1-4and figures 1 and 2). These imbalances are associated with ALL and CML, which are specific cancers. Bentz teaches BCR-ABL positive ALL patients have a poor prognosis and proper detection of the BCR-ABL phenotype allows treatment for this abnormality (see page 1927, column 1, lines 21-23).

Therefore, it would have been prima facie obvious to one of ordinary skill in art at the time the invention was made to use the method of claim 1 of '997 patent to detect chromosomal imbalances and correlate them to ALL and CML as taught by Bentz. The ordinary artisan would be motivated to detect chromosomal imbalances associated with ALL and CML because it would allows directed treatment of this translocation, as taught by Bentz.

Response to Arguments

13. The response filed 1/16/2007, asserts on pages 13-14, that no motivation to combine the cited references is cited.

Applicant's arguments have been fully considered but are not found persuasive.

The office action mailed 9/15/2006 teaches on the last paragraph of page 11 through page 12," the ordinary artisan would be motivated to detect chromosomal

imbalances associated with ALL and CML because it allows directed treatment this translocation." The office action specifically points to page 1927, 1st column, lines 21-23, where Bentz teaches stratification of patients on BCR-ABL phenotype allows directed treatment for this abnormality. Thus motivation for combination is cited.

Summary

No claims are allowed over prior art cited.

Conclusions

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Steven C. Pohnert whose telephone number is 571-272-3803. The examiner can normally be reached on Monday-Friday 7:00-3:30.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla can be reached on 571-272-0735. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

J. Goldberg
3/17/07

Steven Pohnert